949-29 Predictive Value of Exercise Testing for the Detection of Isolated or Non-isolated Left Main Coronary Artery Stenosis

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To evaluate exercise test results and coronary anatomy in patients referred for the evaluation of chest pain, we reviewed the coronary angiograms and exercise tests (Bruce protocol) of 2813 consecutive patients. All patients underwent symptom limited exercise testing two days prior to cardiac catheterization. Left main coronary stenosis (LMCS) was present in 121 patients (pts): 34 had isolated LMCS (group 1), while 87 had concomitant coronary artery disease (group 2). Matched control pts with 3-vessel disease (group 3) had significantly less exertional hypotension i.e. <20 mmHg increase in systolic blood pressure (12%) compared to group 1 (22%) and group 2 (24%, $p < 0.01$), longer exercise duration (6.5 ± 1.4 min) compared to group 1 (4.4 ± 1.2 min, $p < 0.001$) and group 2 (4.1 ± 1.1 min, $p < 0.001$) and less exercise induced atrial arrhythmias (3.1%) compared to group 1 (8.0%, $p < 0.001$).

In addition, termination of the test because of signs of cerebral hypoperfusion (pre-syncope or dizzyness) was present in 3% in group 1 and 5% in group 3, p = ns, compared to 18% in group 2 ($p < 0.001$). From electrocardiographic abnormalities, only one (ST-segment depression ≥1 mm in ≥7 leads including ST-segment elevation in V2) was different in pts with LMCS (18% in group 1, 22% in group 2) compared to pts in group 3, (1%, $p < 0.001$). Multivariate analysis revealed that signs of cerebral hypoperfusion, exertional hypotension and atrial arrhythmias were the 3 independent predictors of LCMS; clinical signs of cerebral hypoperfusion is suggestive for LMCS in addition to concomitant coronary artery disease.

949-30 Cost Effectiveness Analysis of Stress Myocardial Perfusion Imaging in Stable Angina Patients: Influence of Age and Pretest Risk of Coronary Disease

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Cost effectiveness analysis (CEA) evaluates the outcomes and costs of interventions designed to improve health. Cost effectiveness data for stress myocardial imaging in 9,791 stable angina patients (35% female, 42% reversible defects) enrolled from 7 hospitals (annual cardiac death rate = 0.9% over 2.5 ± 1.5 years) was obtained from each hospital using a top-down costing approach, adjusting Medicare charges by cost-charge ratios + Medicare fee schedule for physician costs. CEA = ΔCost/Δlife expectancy from United States population-based tables (cost discount rate = 5%). The table below indicates CEA for clinically low (≤ 15%), intermediate (16-60%), and high (> 60%) risk patients (reference case age = 60 years).

Pretest Cardiac Risk (n)	Annual Cardiac Death	CEA	
		All	Elderly
Low (826)	0.6%	$254,209	$34,650
Intermediate (3,388)	1.6%	$28,887	$23,310
High (1,607)	4.4%	$52,960	$42,737

In classifying CEA by age deciles, a standardized CEA threshold of <$50,000 was achieved for low and high risk patients aged ≥70 years.

Conclusion: Use of stress myocardial perfusion imaging is most cost effective in intermediate risk patients who present with stable angina symptoms or for those who are elderly.

950 Advances in Our Understanding of Aortic Stenosis

Monday, March 17, 1997, 3:00 p.m.–5:00 p.m.
Anaheim Convention Center, Hall E
Presentation Hour: 3:00 p.m.–4:00 p.m.

950-155 Clinical, Echocardiographic and Exercise Predictors of Outcome in a Prospective Study of Valvular Aortic Stenosis

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In 123 adults (mean age 63 ± 16 years) with asymptomatic aortic stenosis (AS), annual clinical, echocardiographic and exercise data were obtained prospectively over a mean follow-up of 2.5 ± 1.4 (range 0.3 to 6.3) yrs. AS-jet velocity (V_{max}) changed by 0.32 ± 0.34 m/s/yr, mean gradient (ΔP) by 7 ± 7 mmHg/yr and valve area (AVA) by −0.12 ± 0.19 cm^2/yr. Kaplan Meier event-free survival, with endpoints defined as death (n = 8) or aortic valve replacement (AVR) (n = 48), was 93 ± 5% at 1 year, 62 ± 8% at 3 years and 26 ± 10% at 5 years.

Univariate predictors of AVR or death included baseline V_{max} (3.9 ± 0.5 vs 3.3 ± 0.5 m/s, $p < 0.001$), mean ΔP (36 ± 12 vs 25 ± 8 mmHg, $p < 0.001$), AVA (1.11 ± 0.34 vs 1.53 ± 0.53 cm^2, $p < 0.001$), and the rate of increase in V_{max} (0.45 ± 0.42 vs 0.23 ± 0.22 m/s/yr p = 0.001), but not age, gender or etiology of AS. Those with an endpoint had a smaller exercise increase in AVA (0.05 ± 0.26 vs 0.27 ± 0.36 cm^2, p = 0.001), blood pressure (15 ± 7 vs 29 ± 20 mmHg) and cardiac output (5.9 ± 4.2 vs 8.5 ± 4.7, p = 0.004), and a greater exercise decrease in stroke volume (−12 ± 20 vs −2 ± 23 ml, p = 0.03).

On Cox regression analysis, multivariate predictors of clinical outcome were V_{max} at baseline ($p < 0.0001$), the rate of change in V_{max} ($p < 0.0001$) and functional status score (p = 0.002). For those with $V_{max} > 4.0$ m/s, the likelihood of remaining alive without AVR at 2 years was only 21 ± 18%, compared to 66 ± 13% for a V_{max} of 3.0–4.0 m/s and 84 ± 16% for a V_{max} < 3.0 m/s ($p < 0.0001$).

These data will be helpful in management and follow-up of adults with asymptomatic AS and will be useful in designing future trials of interventions to slow disease progression.

950-156 Flow-Dependence of Aortic Stenosis Severity: Comparison of the Gorlin and Continuity Equations with Direct Planimetry of the Anatomical Orifice during Transesophageal Echocardiography

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Increase in aortic valve area (AVA) with increasing flow rates is common when using the Gorlin and the continuity equations for AVA calculation. To test whether these changes reflect true increases in anatomical area or inaccuracies in calculation methods, right heart hemodynamics (7 Fr Swan Ganz), cardiac output (thermodilution), transaortic flow velocity (CW Doppler) and direct planimetry of the anatomical orifice (TEE) were obtained simultaneously in 7 patients with isolated aortic stenosis (AVA: 0.63 ± 0.14 cm^2), at baseline and during infusion of 10 μg/kg/min of dobutamine. Infusion of dobutamine resulted in significant increases in heart rate (from 81 ± 12 to 91 ± 22 bpm), cardiac output (from 3.4 ± 1.9 to 5.3 ± 2.7 L/min), transaortic flow rates (159 ± 36 to 249 ± 45 mL/s) and mean pressure gradient (from 33 ± 17 to 50 ± 28 mmHg). As expected, AVAs calculated with both the Gorlin (+29%, from 0.65 ± 0.14 to 0.84 ± 0.16 cm^2, $p < 0.02$) and the continuity (+30%, from 0.60 ± 0.13 to 0.78 ± 0.17 cm^2, $p < 0.03$) equations increased with increasing flow rates. No significant changes were noted, however, in AVA measured by planimetry (from 0.83 ± 0.22 to 0.81 ± 0.23 cm^2, p = ns). At low flow rates, TEE AVA correlated weakly with that calculated with the Gorlin (r = 0.56) or the continuity (r = 0.56) equations. Correlations improved at high flow rates (r = 0.77 and 0.79, respectively). Thus, the data indicate that AVAs calculated with the Gorlin and the continuity equations increase with flow rates, while true anatomical orifice area does not. As calculated AVAs underestimate the true anatomical area at low flow rates, their flow-dependence probably reflects flow-dependent changes in the coefficient of orifice contraction.

950-157 Changes In Coronary Blood Flow In Relation To Changes In Hemodynamic Parameters During Beta Blockade In Patients With Aortic Valve Stenosis

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Alterations in coronary flow velocity (CFV) have been demonstrated at rest in patients with aortic valve stenosis (AVS) but have never been studied after beta-adrenoreceptor blocker administration. Twenty-three patients with significant pure AVS (14 with exertional symptoms [group 1], 9 asymptomatic [group 2] and 8 control subjects [group 3], all with normal coronary arteries, were studied successively at rest, after esmolol infusion (bolus dose of 500 μ/kg/min and by continuous infusion of 50 μg/kg/min) and after atropine administration (0.025 to 0.040 mg/kg) by proximal left anterior descending (LAD) intracoronary Doppler flow velocimetry concomitant with hemodynamic measurements. In all patients, cardiac index increased after atropine but it decreased after esmolol. Systolic retrograde CFV was recorded only in